Angiotensin-(1-7) and Bradykinin in Baroreceptor Reflex Sensitivity in Hypertension

To the Editor:

We read with great interest the article by Dr Nautiyal et al1 dealing with the relationship between angiotensin (Ang)-(1-7) and vagal function in hypertension. The results of their study demonstrated that intracerebroventricular administration of Ang-(1-7) enhanced vagal components of the baroreceptor reflex sensitivity (BRS) for control of heart rate and heart rate variability, independent of altering blood pressure in hypertensive (mRen2)27 rats. Ang-(1-7) did not alter circulating metabolic hormones, nicotinamide adenine dinucleotide phosphate (NADPH) oxidase, or mitogen-activated protein kinase. In addition, tempol, a reactive oxygen species scavenger, had no effect on blood pressure and indices of the baroreceptor functions but significantly reduced NADPH oxidase in the brain of the hypertensive rats. The Ang type 1 receptor blocker, candesartan, normalized blood pressure but did not correct the sympathovagal imbalance. The authors proposed that improvement of vagal function by Ang-(1-7) might be independent of either mitogen-activated protein kinase or NADPH-derived reactive oxygen species in the central nervous system of hypertensive (mRen2)27 rats.

Evidence indicates that several vasoactive substances might actively participate in the regulation of BRS.2,3 It was demonstrated that Ang-(1-7) and bradykinin can interact to modulate baroreflex control of the heart rate, suggesting that the centrally modulatory effect of Ang-(1-7) on the baroreflex might be mediated, at least in part, by the release of kinins.2 Gironacci et al4 demonstrated that Ang-(1-7) decreased K⁺-induced norepinephrine release in the hypothalamus of spontaneously hypertensive rats by the nitric oxide and bradykinin B2 receptor–dependent mechanisms. It can be speculated that the interactions between Ang-(1-7) and bradykinin might have a crucial role in the central modulation of BRS. However, in a study presented earlier, we showed that bradykinin significantly increased the stimulation-evoked norepinephrine release in the hypothalamus of spontaneously hypertensive rats via the dihydropyridine-sensitive calcium channels.5 Therefore, we would like to know whether bradykinin might have synergistic or antagonistic effects on the Ang-(1-7)–induced BRS alterations in hypertensive (mRen2)27 rats in the study by Dr Nautiyal et al. It would be important to assess more precisely the interactions between Ang-(1-7) and bradykinin in the modulation of BRS and their therapeutic potential in the treatment of hypertension.

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Disclosures

None.

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